surface of the skin. It must be understood that this membrane was quite distinct from the amnion.

The epitrichium, therefore, is present both in the Lemurs and in the Sloths, but in the former it does not, after the hairy coat is developed, form a complete envelope for the fœtus, but is broken up before the termination of the period of gestation into more or less detached flakes of membrane.

III. "Note on the Coagulation of the Blood." By L. C. Wooldridge, M.D., M.R.C.P., Co-Lecturer on Physiology at Guy's Hospital. Communicated by Professor Victor Horsley, F.R.S., &c. (From the Laboratory of the Brown Institution.) Received May 24, 1888.

In a paper read before the Royal Society, April 26th, 1888, Dr. Halliburton offers some criticism of my views respecting the coagulation of the blood. In this note I shall briefly summarise and traverse the objections Dr. Halliburton raises to my theory and experiments.

I. Dr. Halliburton suggests that the substance I call A-fibrinogen —which I obtained by cooling peptone-plasma—is not a normal constituent of the blood plasma, but that it is a precipitate of a hemi-albumose, supposed by him to be present in the peptone which is injected into an animal for the purpose of obtaining peptone plasma. I do not use Witte's peptone, as Dr. Halliburton appears to have done, on account of its recognised impurity, but that obtained from Dr. Gruebler's well-known laboratory in Leipsic. This peptone is prepared according to Henniger's method. A 10 per cent. solution of it in $\frac{1}{2}$ per cent. solution of sodium chloride is quite clear after filtration.

It gives no precipitate on cooling to zero.

It disappears wholly from the blood within one or two minutes after injection.

Finally, A-fibrinogen has properties absolutely different from the peptone injected.

Dr. Halliburton appears to think that this substance, A-fibrinogen, exists only in peptone plasma.

I stated in a paper read before the Royal Society in 1885, "On a New Constituent," &c., that it was also present in salt plasma, and I gave details concerning it in the Croonian MS., which is in the archives of the Royal Society. I explained at length in the paper referred to by Dr. Halliburton, and published in Ludwig's 'Festschrift,' 1887, why there are, as has long been known, two varieties of salt plasma, namely, one containing, as I showed, no A-fibrinogen, this being not

spontaneously coagulable, the other containing it, and therefore being spontaneously coagulable.

II. Dr. Halliburton further asserts, that whereas in the abstract of the Croonian Lecture, I described a body, B-fibrinogen, in the paper in Ludwig's 'Festschrift,' published shortly afterwards, this body was not mentioned, or had become identical with the fibrinogen of Hammarsten. This statement is totally incorrect, for on page 228 of Ludwig's 'Festschrift' there will be found a paragraph headed "B-fibrinogen," and on the following page this passage occurs: "Man sieht also dass das Fibrinogen von Hammarsten in Plasma einen Vorgänger hat, welche andere Eigenschaften besitzt, und ich bezeichne diese Substanz als 'B-fibrinogen.'" The differences between the two bodies here referred to are precisely those mentioned in the abstract of the Croonian lecture, and are shortly as follows:—

(*a*.) B-fibrinogen does not clot with fibrin ferment, but it does clot with leucocytes and other animal and vegetable cells.

(*b*.) It clots with substances which can be obtained from these animal and vegetable cells in large quantities, by extraction with water. These substances I call tissue fibrinogens.

(*c*.) It further clots with lecithin.

Hammarsten's fibrinogen, in remarkable contrast with the properties of this body, does not clot with leucocytes or other animal or vegetable cells, nor does it clot with the substances called tissue fibrinogens nor with lecithin.

I would here add that the fibrinogen in most transudation fluids is similar to Hammarsten's fibrinogen. I have clearly indicated these differences in previous publications.

III. With regard to Dr. Halliburton's remark on the relation of lecithin to clotting, I may say that it not only gives rise to clotting in peptone plasma and cooled plasma, but in a solution of fibrinogen isolated from salt plasma and in the plasma obtained from the blood after the injection of tissue fibrinogen. In discussing the experiments on the behaviour of cooled blood towards lecithin, Dr. Halliburton does not recount the details of the experiments, and hence he conveys a misleading impression of the same. It is necessary for these experiments to use a finely particulate and yet thick emulsion of lecithin, for the following very obvious reasons. The lecithin is insoluble in the salt solution into which the blood is received, and a large quantity of blood being received into a relatively small quantity of the salt solution, the lecithin does not come into contact with all the plasma unless a fine thick emulsion be used.

The fact that fluids free from lecithin produce clotting, in no way disproves the contention that lecithin is an essential factor in coagulation, since every variety of fibrinogen contains lecithin. Lecithin

is, next to proteid, the most widely distributed substance in the animal organism. As Hammarsten has well said, "it has been found wherever it has been looked for." Whenever I have stated that lecithin is present in any fibrinogen, I have prepared it and tested for it in the way I have previously repeatedly described in the papers Dr. Halliburton quotes.

IV. The criticisms which Dr. Halliburton passes upon my discovery that tissue fibrinogens cause intravascular clotting when injected into the living circulation, can hardly be regarded seriously; for he asserts that the tissue fibrinogen is a slimy mass, and causes clotting by mechanically plugging the vessels, whereas if he had repeated my experiments he would have found (1) that the fibrinogen is not at all slimy, and (2) that it can hardly be supposed to cause clotting mechanically, since it passes through the right heart, then the capillaries of the lungs, next the left heart and aorta, and finally the capillaries of the alimentary canal before it first causes clotting, *i.e.*, in the portal vein in the dog.

IV. "Note on the Volumetric Determination of Uric Acid." By A. M. Gossage, B.A., Oxon. Communicated by Professor J. Burdon Sanderson, F.R.S. Received May 29, 1888.

Dr. Haycraft has recently proposed a method for the volumetric determination of uric acid in urine ('Brit. Med. Journ.,' 1885, 2, p. 1100) which has great advantages over all former methods in that it is much quicker and easier to manage. The uric acid from 25 c.c. of urine is precipitated by silver nitrate after previous addition of sodium carbonate (to prevent reduction) and ammonia (to dissolve silver chloride, &c.); this precipitate is then collected, washed, and dissolved in nitric acid, and the amount of silver present in this solution ascertained by Volhard's method, *i.e.*, titration with ammonium sulphocyanate; from this the amount of uric acid can be calculated. "In order to test the accuracy of the process," he says, "I prepared several solutions of acid urate of sodium of known strength. To these I added various quantities of common salt, magnesium sulphate, and phosphate of soda in order to imitate as far as possible the urinary secretion. On estimating the uric acid in these solutions, I obtained wonderfully correct results. In all cases not much more than a milligramme was lost during the process, and may be simply accounted for by the fact that no salt of uric acid is absolutely insoluble. . . . . In order further to test its accuracy, 50 c.c. of urine were divided into two equal portions; to the first 25 c.c. of a solution of acid urate of sodium of known strength were added; to